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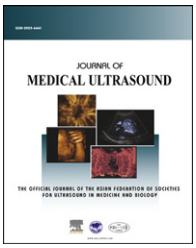

ORIGINAL ARTICLE

Changes of Left Atrial Phasic Function Assessed by Speckle Tracking Echocardiography in Untreated Hypertension

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Changes in the three phases of left atrial (LA) function have not been well studied in hypertension. We studied phasic function of LA by speckle tracking echocardiography (STE) in hypertension. Sixty-one consecutive untreated hypertension patients and 32 sex- and age-matched healthy participants were included. Two-dimensional (2D) images obtained from apical four-chamber and two-chamber views were used for STE analysis of LA. LA peak positive strain (39.8 ± 10.9 vs. 45.3 ± 12.7 %, $p = 0.031$), peak negative strain (−2.2 ± 1.5 vs. −3.0 ± 1.9 %, $p = 0.029$), and conduit strain rate (−2.0 ± 0.5 vs. −2.4 ± 0.6 1/s, $p = 0.005$) were significantly decreased in hypertension. Twenty hypertensive patients (32%) had diastolic dysfunction. Positive strain (34.8 ± 7.2 vs. 42.2 ± 11.6 %, $p = 0.012$) and conduit strain rate (−1.7 ± 0.4 vs. −2.2 ± 0.5 1/s, $p < 0.001$) were decreased in patients with diastolic dysfunction. Only conduit strain rate was independently associated with diastolic dysfunction (OR 11.9, $p = 0.026$) after multivariable analysis. LA phasic function is decreased in untreated hypertension. Decreased LA conduit function is associated with diastolic dysfunction in hypertension.

Introduction

Hypertension is high in prevalence and has many adverse effects on the heart. Changes in the left ventricle (LV) under the effects of hypertension are well studied

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including LV hypertrophy, abnormal geometry, and diastolic dysfunction [1]. The left atrium (LA) is also affected by hypertension and LA enlargement is noted even earlier in mild hypertension [2]. Increased LA size is associated with risk of developing atrial fibrillation and stroke [3]. Although LA enlargement is well known as an important change for hypertensive heart disease, however, changes of LA function, especially in different phases, have not been fully elucidated.

LA volume and function measured by two-dimensional (2D) biplane area length and real-time 3D echocardiography have been well validated and widely used. LA function consists of three components, including reservoir, conduit, and active contractile phases [4]. The most frequently used echocardiographic method for evaluation of phasic function of LA is the volumetric method [5]. LA volume measured by the biplane area-length method and corrected by body surface area is a useful index for studying LA remodeling, and early LA remodeling has been proven to have prognostic significance in heart failure after myocardial infarction [6]. Phasic changes in LA volume can be measured in different phases of the cardiac cycle. LA total emptying fraction (LATEF), representing the total function of the LA, passive emptying fraction (LAPEF), representing LA conduit function, and active emptying fraction (LAAEF), representing LA active pumping, can all be measured from phasic changes of LA volume [7]. LA active pumping volume and LAAEF have been found increased in mild hypertension [2]. Volumetric measurements are useful in evaluation of LA, however, volumetric changes cannot reflect changes in the LA wall itself.

Tissue Doppler imaging has been applied for the assessment of cardiac function, including atrial function [8]. Atrial deformation evaluated by tissue Doppler-derived strain or strain rate imaging can be used for assessment of LA wall directly [9]. The newly developed speckle tracking echocardiography (STE) is useful in the assessment of left ventricular strain and strain rate [10]. This non-Doppler 2D strain imaging avoids the effects of Doppler angle and ventricular tethering [11]. STE was recently used for assessment of LA function after resynchronization therapy [12]. The feasibility of LA longitudinal strain and strain rate measured by STE has been verified in healthy individuals, and adequate tracking can be performed in 94% of individuals or in 97% of segments [13,14].

A recent study has demonstrated that the subtle deterioration of LV systolic function can be detected by decreased longitudinal strain from speckle tracking in diastolic heart failure [15]. We hypothesized that early changes in LA function in hypertension can be documented by STE. The purpose of this study was to evaluate the phasic LA function with untreated hypertension individuals and correlate with LV diastolic function.

Methods

Participants

This study recruited 64 consecutive patients with untreated hypertension in a hypertension clinic. Three patients were excluded due to inadequate images and the remaining 61 participants (mean age 57 $\pm$ 13 years, 31 men) formed the study group. Thirty-two age- and sex-matched healthy individuals (mean age 53 $\pm$ 11 years, 13 men) from health examinations were recruited as a control group. Untreated hypertension was diagnosed if blood pressure was $\geq$140/90 mmHg on three occasions and the patient was not receiving any antihypertensive treatment in the recent 6 months. The normal group was defined as individuals without any cardiovascular diseases and risk factors who were normal in physical examination and echocardiogram. Patients were excluded if they had atrial fibrillation, significant valvular heart diseases, or poor imaging quality. The study was approved by the research ethics committee of our hospital, and informed consent was obtained from all individuals.

Echocardiography

Standard echocardiography was performed with Doppler studies (Vivid 7, GE-VingMed, Horten, Norway) with a 3.5-MHz multiphase array probe in individuals lying in left lateral decubitus position. The chamber dimension and wall thickness were measured by 2D guided M-mode method and LV ejection fraction was measured by the 2D biplane method of discs, according to the recommendations of the American Society of Echocardiography [16]. Left ventricular mass was measured by M-mode method and indexed by body surface area [17]. Transmitral Doppler flow velocity was obtained from apical four-chamber view, and peak early filling velocity (E), peak atrial velocity (A), early filling-to-atrial velocity ratio (E/A), and mitral deceleration time (DT) were measured [18]. Pulse tissue Doppler imaging was obtained; early diastolic annulus velocity (E') and atrial annulus velocity (A') were measured at both the septal and lateral annulus [19]. Diastolic dysfunction was diagnosed if either medial E/E' $\geq$ 15 or lateral E/E' $\geq$ 12 with abnormal mitral inflow pattern (E/A $<$ 1 or $\geq$ 1 with DT $\leq$ 140 ms) [20]. 2D images were acquired from apical four-chamber and two-chamber views for three cardiac cycles and digitally stored with a frame rate of 50—90 frames per second. The images were analyzed offline by computer software (EchoPac PC 08, GE-VingMed, Horten, Norway).

Volumetric measurements of LA

The LA volume was measured by the biplane area-length method from 2D echocardiography [21]. The LA area was measured with a planimetry for four-chamber and two-chamber views by tracing the endocardial border, excluding the confluence of the pulmonary veins and the LA appendage. The LA length was measured from the midline of the plane of the mitral annulus to the opposite aspect of the LA. LA volume was measured at end-systole, before P-wave, and end-diastole, calculated as 0.85 $\times$ four-chamber area $\times$ two-chamber area / average of the two lengths [21]. Four phasic LA emptying fractions were used as indexes for LA function including LATEF for total function, LA expansion index (LAEI) for reservoir function, LAPEF for conduit function, and LAAEF for active pumping [7]. LA size was represented by LA maximal volume measured at end-

systole and indexed by body surface area (LA volume index, LAVI). Measurements were repeated three times in each individual, and the average was used for analysis.

Measurements of LA deformation by STE

The endocardial border of the LA was manually defined using a point-and-click technique. An epicardial surface tracing was automatically generated by the system, creating a region of interest, which was manually adjusted to cover the full thickness of the myocardium in the systolic frame. The width of the smallest region of interest was 8 mm. Before processing, a cine loop preview was used to confirm if the internal line of the region of interest followed the LA endocardial border throughout the cardiac cycle. Time-strain and time-strain rate plots were produced automatically by the software. Peak positive LA strain (LASp) during left ventricular filling and peak negative strain (LASn) after P-wave were measured from the strain curve. Peak positive filling strain rate (LASRf) during LA filling, peak negative conduit strain rate (LASRc) in left ventricular early filling, and peak negative atrial strain rate (LASRa) after P-wave were identified from the strain rate curve (Fig. 1). We further divided the LA wall into eight segments including basal septal, middle septal, basal lateral, and middle lateral segments on four-chamber view and basal inferior, middle inferior, basal anterior, and middle anterior segments on two-chamber view. The average of LASp, LASn, LARSf, LASRc, and LASRa of eight segments were used for analysis. In our echocardiography laboratory, intra-observer and inter-observer variability for LA strain were 6.8% and 8.9%, and for LA strain rate 3.3% and 6.2%, respectively [22].

Statistics

Differences between control and hypertension groups were compared with Student *t* test, for continuous variables, or the Chi-square test, for categorical variables. Then we analyzed the differences between hypertensive patients with or without diastolic dysfunction by using Student *t* test, for continuous variables, or the Chi-square test, for categorical variables. Multiple logistic regression analysis was used for independent factors for diastolic dysfunction in the hypertensive group. Correlation between strain and volumetric parameters were analyzed by using Pearson's correlation test. All data are presented as the mean $\pm$ standard deviation (SD). A p value of less than 0.05 was considered statistically significant. All analysis was performed with SPSS 12.0 for Windows (SPSS Institute, Chicago, IL, USA).

Results

Differences between hypertension and control

The comparison of basic data between patients with hypertension and normal controls is listed in Table 1. There were no differences in age and sex. Hypertensive patients had higher wall thickness, LV mass index, relative wall thickness, and LA volume index (LAVI) but not LV size and ejection fraction (Table 1). For the diastolic parameters, hypertensive patients had higher E, lower E/A, lower E' at both sides of the annulus, and higher E/E' at both sides of the annulus (Table 1). For volumetric measurements of LA, hypertensive patients had increased LA volume in all three phases, however, only total empting volume and active emptying volume were higher in hypertensive patients but not emptying fraction of any phase (Table 2). For deformation of LA, LASp, LASn, and LASRc were significantly decreased in those with hypertension (Table 2). By using the method previous reported [23], calculated LA systolic force (LASF) was significantly increased in the hypertension group (Table 2).

Factors contributed to diastolic dysfunction in hypertension

There were 20 patients (33%) in the hypertension group with diastolic dysfunction and seven of them had heart failure symptoms. Comparing hypertensive patients with or without diastolic dysfunction, age was higher in patients with diastolic dysfunction (Table 3). A was higher and E' of both mitral annulus was lower in diastolic dysfunction (Table 3). LA passive emptying volume and LAPEF were decreased, and LAAEF was increased in hypertensive patients with diastolic dysfunction (Table 4). LASF was increased in diastolic dysfunction. LASp and LASRc were the only deformation parameters decreased in diastolic dysfunction (Table 4). After stepwise multiple logistic regression analysis controlling for factors that were generally believed would affect diastolic function (LAVI, LV mass index, systolic blood pressure) or significant different in single variable analysis (age), only LASRc was independently associated with diastolic dysfunction in hypertension (Table 5). Comparing patients with or without heart failure symptoms (greater than New York Heart Association Function Class I) in diastolic dysfunction, LASRc was still significantly lower in patients with heart failure symptoms (-1.40 ± 0.33 vs. -1.85 ± 0.28 1/s, $p = 0.004$).

Correlation between volumetric parameters and deformation of phasic atrial function

Parameters of LA deformation from STE were correlated with corresponding phasic volumetric parameters. By adjusting age and LA volume index, LASRf was significantly correlated with LAEI (Beta $= 0.419$, $p < 0.001$), LASRc with LAPEF (Beta $= -0.470$, $p < 0.001$), LASRa with LAAEF (Beta $= -0.383$, $p < 0.001$), and LASp with LATEF (Beta $= 0.418$, $p < 0.001$).

Discussion

Our present study has demonstrated that LA function measured by LA deformation was changed in patients with untreated hypertension. Decreased passive or conduit function and increased active function of LA and LASF contributed to diastolic dysfunction in hypertension. However, LASRc was the only parameter among the

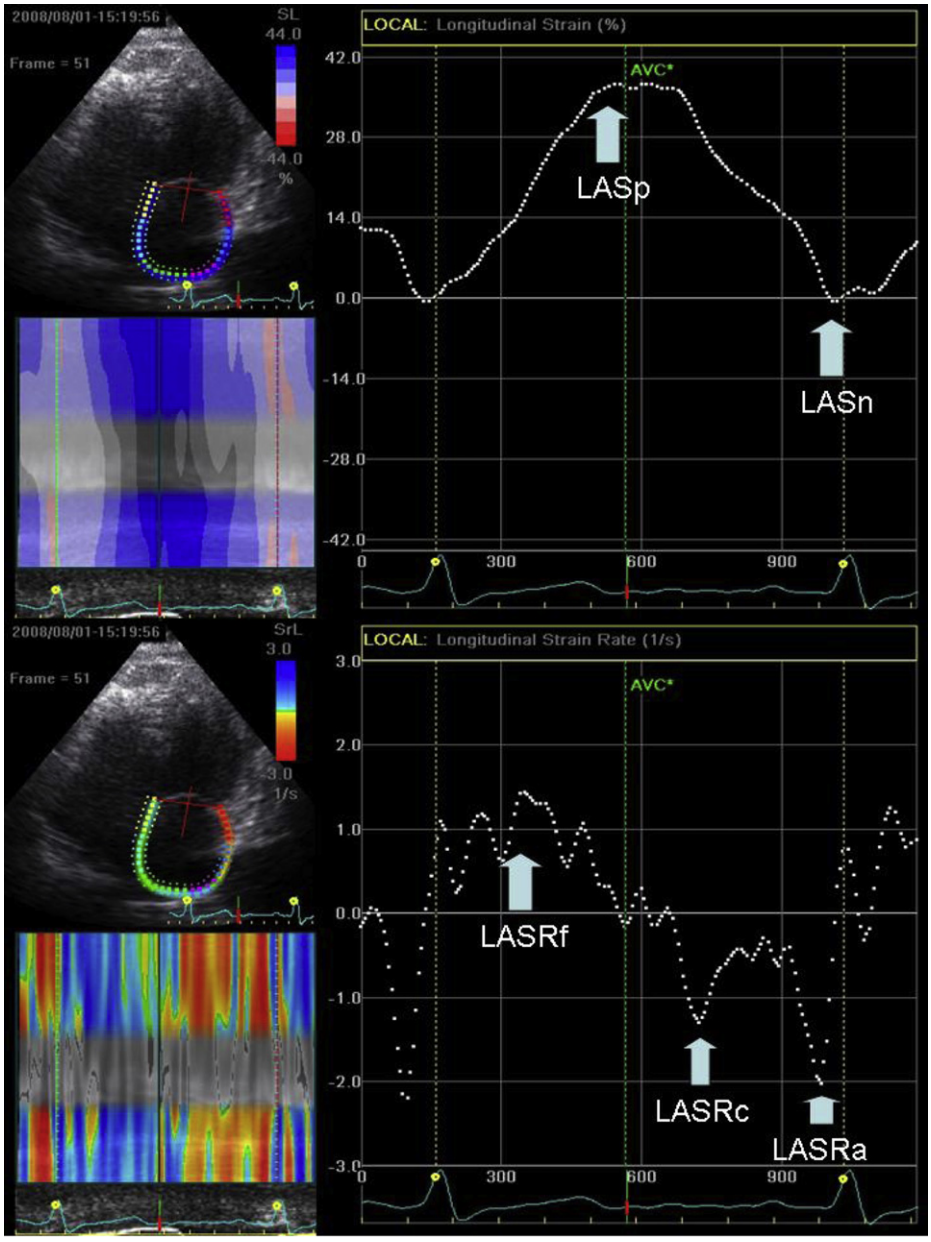

Fig. 1 The figure shows strain (upper panel) and strain rate (lower panel) curves of the left atrium (LA) by using speckle tracking echocardiography (STE). LASp represents left atrial peak positive strain, and LASn represents left atrial peak negative strain after P-wave. LASRf represents left atrial peak positive strain rate in left atrial filling, LASRc represents left atrial peak negative strain rate in left ventricular early filling, and LASRa represents left atrial peak negative strain rate during atrial contraction.

volumetric parameters and LA deformation independently associated with diastolic dysfunction.

Cardiac structural and functional changes in hypertension

As a direct target organ of hypertension, LV hypertrophy is the most pronounced change in the heart and it is associated with increased cardiovascular risk [24]. Hypertension also causes changes in LV geometry, decreased myocardial deformation, and LV diastolic dysfunction [1,24—26]. The effects of hypertension on LA have not been thoroughly studied. LA enlargement is associated with LV geometry and diastolic heart failure [2,27]. Part of our study results were compatible with other studies [2,23]. LV mass index, LA volume index, and LASF were all increased suggesting that hypertensive heart disease is involved in both LA and LV early in untreated hypertension. Unique findings from changes of LA deformation in our study added important evidence that changes of LA in hypertensive heart disease was not only a compensatory response to LV.

Changes in phasic function of LA in hypertension

In our study, all of the LA volumes in different phases were increased in hypertension. Total emptying volume and active emptying volume were increased, but all of the LA emptying fractions were not changed in hypertension.

Table 1 Comparison of basic data and echocardiographic parameters between hypertension and normal control.

	Hypertension ($n = 61$)	Normal ($n = 32$)	p
Age (years)	57 ± 13	53 ± 11	0.157
Male (%)	31 (51)	13 (41)	0.350
Body weight (kg)	66 ± 14	60 ± 10	0.013
Body height (cm)	159 ± 10	159 ± 9	0.972
SBP (mmHg)	158 ± 21	121 ± 13	<0.001
DBP (mmHg)	94 ± 16	71 ± 10	<0.001
Septal wall thickness (cm)	0.97 ± 0.22	0.76 ± 0.17	<0.001
LV end-diastolic diameter (cm)	4.67 ± 0.50	4.74 ± 0.49	0.516
Posterior wall thickness (cm)	0.97 ± 0.22	0.75 ± 0.16	<0.001
LV end-systolic diameter (cm)	2.68 ± 0.47	2.71 ± 0.37	0.758
LV ejection fraction (%)	73 ± 9	74 ± 6	0.751
LVMI (gm/m^2)	91.79 ± 29.29	70.60 ± 18.23	<0.001
Relative wall thickness	0.42 ± 0.10	0.32 ± 0.07	<0.001
LAVI (ml/m^2)	23.89 ± 7.26	19.42 ± 4.88	0.001
E (cm/s)	66.6 ± 16.4	69.2 ± 17.9	0.487
A (cm/s)	82.3 ± 15.7	68.2 ± 16.6	<0.001
E/A	0.83 ± 0.26	1.08 ± 0.40	0.004
DT (ms)	192.3 ± 51.9	192.8 ± 44.6	0.964
Septal E' (cm/s)	6.6 ± 2.3	8.8 ± 3.1	0.001
Septal A' (cm/s)	9.7 ± 1.9	9.4 ± 1.8	0.514
Lateral E' (cm/s)	8.3 ± 2.9	10.7 ± 3.5	0.001
Lateral A' (cm/s)	9.8 ± 2.5	9.4 ± 2.2	0.425
Septal E/E'	11.1 ± 3.8	8.7 ± 3.4	0.005
Lateral E/E'	8.7 ± 2.7	7.1 ± 2.5	0.011

Data are expressed as mean ± SD or number (%).
A = atrial velocity of mitral flow; A' = atrial velocity of mitral annulus; DBP = diastolic blood pressure; DT = deceleration time of E; E/A = E-to-A ratio; E/E' = E-to-E' ratio; E = early diastolic velocity of mitral flow; E' = early diastolic velocity of mitral annulus; LAVI = left atrial volume index; LV = left ventricle; LVMI = left ventricular mass index; SBP = systolic blood pressure.

Table 2 Comparison of volumetric measurements and deformation of left atrium between hypertension and normal control.

	Hypertension ($n = 61$)	Normal ($n = 32$)	p
Volumetric measurements			
LAVs (mL)	40.3 ± 13.3	33.4 ± 10.8	0.013
LAVp (mL)	29.5 ± 10.1	23.2 ± 7.9	0.003
LAVd (mL)	15.9 ± 7.0	12.4 ± 5.5	0.016
LATEV (mL)	24.4 ± 8.3	21.0 ± 5.5	0.048
LAPEV (mL)	10.8 ± 8.3	10.1 ± 6.1	0.694
LAAEV (mL)	13.6 ± 5.1	10.8 ± 4.0	0.009
LATEF (%)	61.2 ± 10.3	63.2 ± 10.9	0.402
LAPEF (%)	25.5 ± 16.8	29.6 ± 14.2	0.240
LAAEF (%)	46.6 ± 11.7	47.6 ± 12.4	0.693
LAEI (%)	177.0 ± 86.7	195.9 ± 91.2	0.329
LASF (kdyn)	13.3 ± 7.0	9.0 ± 6.4	0.005
Deformation indexes			
LASp (%)	39.77 ± 10.90	45.29 ± 12.70	0.031
LASn (%)	−2.18 ± 1.54	−2.99 ± 1.90	0.029
LASRf (1/s)	2.89 ± 0.77	3.01 ± 0.59	0.424
LASRc (1/s)	−2.04 ± 0.51	−2.39 ± 0.62	0.005
LASRa (1/s)	−2.94 ± 0.64	−3.13 ± 0.64	0.168

Data are expressed as mean ± SD.
LAAEF: left atrial active emptying fraction; LAAEF: left atrial active emptying volume = LAVp−LAVd; LAEI: left atrial expansion index; LAPEF: left atrial passive emptying fraction; LAPEV: left atrial passive emptying volume = LAVs−LAVp; LASn: negative strain of left atrium; LASp: positive strain of left atrium; LASR: left atrial systolic force; LASRa: strain rate in left atrial contraction phase; LASRc: strain rate in left atrial conduit phase; LASRf: strain rate in left atrial filling phase; LATEF: left atrial total emptying fraction; LATEV: left atrial total emptying volume = LAVs−LAVd; LAVd: left atrial volume in diastole; LAVp: atrial volume before P-wave; LAVs: left atrial volume in systole.

Unlike volumetric parameters, LA deformation indexes including LASp, LASn, and LASRc were decreased in hypertension. Similarly, our previous study showed that LA deformation was not decreased in the active contraction phase [28]. Decreased LASp represented decreased reservoir function of the LA. Our result was similar to a previous study using tissue Doppler derived strain rate to demonstrate decreased reservoir function of LA in early hypertension [29]. Decreased LA reservoir function was found to be associated with the occurrence of atrial fibrillation [30], and explained partially why hypertension increased the risk for atrial fibrillation. Decreased LASRc was the most pronounced change in LA deformation in this study. Our study also demonstrated E' of both annulus decreased in hypertension. The simultaneous changes in LA conduit and LV diastolic function reflected the increased chamber stiffness due to hypertension that occurred both on the LA and the LV at the same time. A recent study demonstrated a parallel decrease in LV early diastolic strain rate and LA conduit strain rate measured by tissue Doppler imaging in hypertensive diabetes patients [31]. Volumetric parameters in the conduit phase of LA (LA passive emptying volume or LAPEF) were not decreased in our study. Passive emptying volume or fraction did not change because all phases of LA volumes were increased in hypertension in a similar proportion except for the active phase. Compensation for the increased stiffness in LA and LV was by increasing LA active contraction and could be demonstrated by increasing LASF and A of transmitral flow shown in our study and previous studies [1,2,23].

Our study also demonstrated that LAPEF, LASp, and LASRc decreased and LASF and LAAEF increased significantly in hypertensive patients with diastolic dysfunction. All of the findings indicated that deterioration of LA function contributed actively in the development of diastolic dysfunction in hypertension. LA was not only passively changed in response to increased LV filling pressure. Decreased LA strain or increased LA stiffness were more accurate indexes for identifying diastolic dysfunction [27].

Table 3 Comparison of basic data and echocardiographic parameters between patients with and without diastolic dysfunction in hypertension.

	DD+ (n = 20)	DD− (n = 41)	p
Age (years)	65 ± 10	53 ± 12	<0.001
Male (%)	8 (67)	23 (56)	0.238
Body weight (kg)	68 ± 17	65 ± 12	0.558
Body height (cm)	157 ± 11	160 ± 10	0.370
SBP (mmHg)	159 ± 23	158 ± 20	0.891
DBP (mmHg)	93 ± 18	95 ± 15	0.692
Septal wall thickness (cm)	1.07 ± 0.23	0.92 ± 0.20	0.015
LV end-diastolic diameter (cm)	4.70 ± 0.63	4.65 ± 0.43	0.728
Posterior wall thickness (cm)	1.03 ± 0.24	0.93 ± 0.20	0.094
LV end-systolic diameter (cm)	2.68 ± 0.44	2.68 ± 0.49	0.970
LV ejection fraction (%)	74 ± 9	73 ± 10	0.845
LVMI (gm/m^2)	99.84 ± 33.33	87.96 ± 26.77	0.147
Relative wall thickness	0.45 ± 0.11	0.40 ± 0.09	0.056
LAVI (ml/m^2)	22.11 ± 7.79	24.74 ± 6.94	0.198
E (cm/s)	65.5 ± 18.5	67.2 ± 15.5	0.716
A (cm/s)	90.3 ± 15.2	78.4 ± 14.6	0.005
E/A	0.75 ± 0.29	0.88 ± 0.24	0.062
DT (ms)	200.2 ± 53.7	188.4 ± 51.2	0.412
Septal E ± ' (cm/s)	4.8 ± 2.0	7.4 ± 1.8	<0.001
Septal A' (cm/s)	10.2 ± 2.4	9.5 ± 1.6	0.221
Lateral E' (cm/s)	6.0 ± 2.1	9.4 ± 2.5	<0.001
Lateral A' (cm/s)	10.0 ± 2.7	9.8 ± 2.5	0.775
Septal E/E'	14.8 ± 4.0	9.3 ± 2.1	<0.001
Lateral E/E'	11.4 ± 2.7	7.3 ± 1.5	<0.001

Data are expressed as mean ± SD or number (%).
A = atrial velocity of mitral flow; A' = atrial velocity of mitral annulus; DBP = diastolic blood pressure; DD = diastolic dysfunction; DT = deceleration time of E; E/A = E-to-A ratio; E/E' = E-to-E' ratio; E = early diastolic velocity of mitral flow; E' = early diastolic velocity of mitral annulus; LAVI = left atrial volume index; LV = left ventricle; LVMI = left ventricular mass index; SBP = systolic blood pressure.

Table 4 Comparison of volumetric measurements and deformation of left atrium between patients with and without diastolic dysfunction in hypertension.

	DD+ (n = 20)	DD− (n = 41)	p
Volumetric measurements			
LAVs (mL)	37.7 ± 14.2	41.6 ± 12.9	0.293
LAVp (mL)	30.3 ± 9.3	29.2 ± 10.5	0.696
LAVd (mL)	15.1 ± 7.1	16.3 ± 7.1	0.532
LATEV (mL)	22.6 ± 8.5	25.3 ± 8.2	0.249
LAPEV (mL)	7.5 ± 7.6	12.4 ± 8.2	0.028
LAAEV (mL)	15.2 ± 5.5	12.9 ± 4.8	0.100
LATEF (%)	60.9 ± 8.7	61.5 ± 11.1	0.839
LAPEF (%)	17.1 ± 14.5	29.6 ± 16.4	0.005
LAAEF (%)	51.2 ± 13.5	44.4 ± 10.1	0.030
LAEI (%)	175.1 ± 103.1	177.9 ± 79.0	0.907
LASF (kdyn)	17.1 ± 7.4	11.4 ± 6.0	0.002
Deformation indexes			
LASp (%)	34.79 ± 7.20	42.19 ± 11.62	0.012
LASn (%)	−2.38 ± 2.15	−2.09 ± 1.15	0.585
LASRf (1/s)	2.65 ± 0.48	3.00 ± 0.86	0.092
LASRc (1/s)	−1.69 ± 0.38	−2.21 ± 0.49	<0.001
LASRa (1/s)	−2.99 ± 0.59	−2.91 ± 0.66	0.641

Data was expressed as mean ± SD.
DD: diastolic dysfunction; LAAEF: left atrial active emptying fraction; LAAEF: left atrial active emptying volume = LAVp−LAVd; LAEI: left atrial expansion index; LAPEF: left atrial passive emptying fraction; LAPEV: left atrial passive emptying volume = LAVs−LAVp; LASn: negative strain of left atrium; LASp: positive strain of left atrium; LASR: left atrial systolic force; LASRa: strain rate in left atrial contraction phase; LASRc: strain rate in left atrial conduit phase; LASRf: strain rate in left atrial filling phase; LATEF: left atrial total emptying fraction; LATEV: left atrial total emptying volume = LAVs-LAVd; LAVd: left atrial volume in diastole; LAVp: atrial volume before P-wave; LAVs: left atrial volume in systole.

Study limitations

Image quality affected the precision of the measurements. The difficult imaging of the LA using transthoracic echocardiography limited the application of speckle tracking in evaluation of the LA. However, 97% of individuals could be studied in this study indicating that STE for LA was feasible. Although a previous study recommended volumetric measurements for the assessment of LA phasic function in hypertension [32], our study added important information to the understanding of hypertensive heart disease using STE. Together with volumetric measurements, we can evaluate LA more comprehensively and extensively by using deformation imaging. This study was a cross-sectional study and lacked clinical follow-up. The clinical implications of LA mechanical dysfunction should be further studied. Although the analysis in our study was based on echocardiography-defined diastolic dysfunction, patients with heart failure

Table 5 Multivariate logistic analysis of left atrial parameters controlling for age, left atrial volume index, left ventricular mass index, and systolic blood pressure for the independency to diastolic dysfunction.

	Odds ratio	95% Confidence interval	p
LAPEF	0.972	0.927−1.021	0.257
LAAEF	1.068	0.984−1.159	0.117
LASF	1.069	0.964−1.185	0.207
LASp	0.905	0.810−1.011	0.078
LASRc	11.911	1.351−104.979	0.026

LAAEF = left atrial active emptying fraction; LAPEF = left atrial passive emptying fraction; LASF = left atrial systolic force; LASp = positive strain of left atrium; LASRc = strain rate in left atrial conduit phase.

symptoms had significantly lower LASRc. The effects of LA strain or strain rate on heart failure symptoms in hypertensive heart disease need to be documented in further large-scale studies because only a few patients had heart failure symptoms in our study. Finally, the software was developed for assessment of LV and its use for LA assessment has not fully validated. However, the feasibility of STE for LA and its clinical usefulness have been studied [13,14,22,28,33]. We need more studies to validate this technology.

Conclusions

Our study demonstrated that early changes in atrial strain rate in the conduit phase was identified in untreated hypertensive individuals with left ventricular diastolic dysfunction. It may provide further important prognostic parameter in hypertensive patients.

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